Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Critical Care

SESSION TITLE: Medical Student/Resident Critical Care Posters
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CORONA VIRUS DISEASE-19-INDUCED ACUTE LIVER FAILURE LEADING TO SEVERE METABOLIC ACIDOSIS

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INTRODUCTION: 14-53% of coronavirus disease 2019 (COVID-19) patients have elevated alanine aminotransferase(ALT) and aspartate aminotransferase (AST).[1] We present a case of COVID-19 who developed severe metabolic acidosis from acute liver failure.

CASE PRESENTATION: 53-year-old male with a history of cardiomyopathy (Ejection Fraction [EF] of 15%) was admitted with shortness of breath and tested positive for COVID-19. He was hypoxic (2-3 liters on nasal cannula [NC]) and had a blood pressure (BP) of 100/70 mm of Hg. His initial labs were significant for mild elevations in AST and ALT with normal alkaline phosphatase. He underwent CT scan of the chest and abdomen, which showed nonspecific lung nodules and thickened gall bladder. His liver enzymes began to rise serially reaching an AST of 4,735 and ALT of 1,988. He was treated with convalescent plasma on day 2 of admission. Hepatitis panel was negative. Ultrasound arterial doppler of the abdomen and HIDA scan ruled out portal vein thrombosis and cholecystitis, respectively. He developed tachypnea to 50 breaths/minute and respiratory distress. An arterial blood gas showed pH:7.5, pCO2:10, pO2:68, HCO3:10 and a lactate of 16. Initial concerns were for cardiogenic shock (due to low EF) vs sepsis. However, BP was stable with good urine output. He was transferred to the ICU. Repeat echocardiography(TTE) showed an EF of 15-25%. He was placed on broad antibiotics and received N-acetylcysteine (NAC) for liver failure. Repeat infectious exam and imaging were negative. His lactic acidosis improved, and he was downgraded within 48 hours.

DISCUSSION: Normal BP, evidence of good end organ perfusion, unchanged TTE findings and negative infectious work up ruled out cardiogenic and septic shock. Negative hepatitis viral screen, negative imaging tests and absence of hepatotoxic agents ruled out other causes of liver injury. NAC administration coincided with clinical improvement, although its role in the recovery process remained unclear. Severe acute respiratory syndrome coronavirus 2 [SARS-CoV-2] binds to angiotensin-converting enzyme 2 (ACE2) receptor to enter target cells. Studies have shown ACE2 receptors to have increased activity in cholangiocytes through which SARS-COV-2 can cause liver injury [2]. COVID-19 is known to cause multi-organ failure leading to acidosis.However,clinicians must be aware of lactic acidosis from purely COVID-19-induced acute liver failure as seen above.

CONCLUSIONS: Acute onset lactic acidosis can be indicative of multiple processes which can be challenging to interpret in an urgent setting. Respiratory distress in COVID-19 patients is not always due to an acute lung process but may be related to metabolic acidosis induced tachypnea. Future studies looking at use of NAC in COVID-19-induced liver failure may be beneficial.

Reference #1: Zhang, Chao et al. "Liver injury in COVID-19: management and challenges." The lancet. Gastroenterology & hepatology vol. 5,5 (2020): 428-430

Reference #2: Chai X Hu, L Zhang Y et al. Specific ACE2 expression in cholangiocytes may cause liver damage after 2019-nCoV infection. bioRxiv. 2020; (published online Feb 4.)

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